Modern Concepts of Cardiovascular Disease

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THE FUNCTIONAL CONSEQUENCES OF ACUTE CORONARY OCCLUSION

The outstanding phenomena following acute coronary occlusion are: (1) cardiocirculatory failure characterized by hypotension, feeble pulse, venous congestion, dyspnea, cyanosis, pallor, sweating and so forth, (2) irregular or rapid action of the heart and (3) pain usually, but not always, agonizing in character and radiating from the region of the sternum outward to the left arm, upward toward the throat (angina), or downward to the epigastrium; at one time the pain may extend in any or all of the directions named.

Analysis on basis of experimental studies

Cardiocirculatory failure. — Owing to scant development of anastomotic connections between branches of the three main coronary rami and to high resistance against flow of blood through these connections, occlusion of any one ramus in an otherwise normal heart results in ischemia of the region supplied. As a result, contraction in the region of ischemia ceases approximately within a minute after occlusion has taken place; instead of shortening and moving inward, the wall of the affected portion expands and bulges outward, due to the rise of intraventricular pressure occasioned by contraction of unaffected myocardium. The total force for expulsion of blood is thus reduced, not only by loss of function of some of the formerly contracting fractions but also because part of the energy is spent in expanding the noncontracting portion of the ventricular wall. The duration of systole is also reduced somewhat. The immediate effects on the forward movement of blood are: decreased systolic discharge, decreased aortic pressure and a smaller pulse pressure. The effects of back pressure are: dilatation of the ventricles and elevation of pulmonary and venous pressure. Provided the unaffected myocardium is in good responsive condition, compensation is accomplished and normal circulatory balance is restored. Briefly, the sequence of events is this: During initial hypodynamic beats, small unexpelled volumes added to normal filling volumes raise diastolic pressure in the ventricles and stretch the viable muscle. In accordance with the law of initial tension and length, (recently reviewed in these leaflets (Visscher, Maurice B.: Heart failure. Mod. Concepts Cardiovasc. Dis. 14:No. 8 (Aug.) and No. 9 (Sept.) 1945), the viable muscle contracts more vigorously than it did before the diastolic pressure in the ventricles was raised, thereby restoring cardiac output and arterial pressure to normal.

If, however, the viable portion is already damaged functionally, or is operating too near its reserve limit, compensation cannot occur; on the contrary, decompensation may progress even to the point of death. As a result of the lowering of arterial pressure, moderator reflexes over sinus and aortic nerves are released which, together with pain reflexes, cause intense vasoconstriction in the viscera, muscles and skin. Constriction of cutaneous vessels (causing pallor) diverts a considerable volume of blood to internal organs, tends to intensify the engorgement of the viscera and adds to the volume of venous blood that the decompensated myocardium cannot move. The patient appears to be in a state of shock indistinguishable from that caused by loss of blood or plasma, except that an excessive accumulation of blood is found in the internal venous system.

Cardiac irregularities.—It long has been suspected that irritable foci develop at the margins of the ischemic portion of the heart. These give rise to occasional, or frequent, premature ventricular contractions or to transient periods of ventricular tachycardia. Occasionally, these centers emit an accelerating series of impulses which eventuate in ventricular fibrillation when the rate of emission becomes critical. The mechanism appears to resemble that produced by application of a direct current to the normal myocardium. Such a current is known to set up a depolarized condition which leads to spontaneous discharges. Similarly, there is evidence that electrotonic differences in potential are set up between normal and ischemic muscles and that these differences give rise to periodic discharges. When the repetitive discharges occur at an accelerating speed, not all of the resulting impulses pass radially to normal fractions of the heart muscle; some are blocked, owing to the presence of refractory portions and the impulses begin to take a zigzag course, thus giving rise to fibrillation.

Pain.—Impulses which initiate the pain travel over visceral afferent elements of the cardiac sympathetic nervous system, enter the spinal cord by white rami of the first to the sixth thoracic nerves inclusive, and pass upward to the thalamus and cerebral cortex. Connections which are made at various levels in their ascent give rise to (a) viscero-motor reflexes (increased tonus of thoracic muscles, sense of constriction of chest or throat), (b) viscero-visceral reflexes to the stomach (reduction of tonus, reverse gastric peristalsis and so forth, leading to sinking feeling in the epigastrium, nausea, vomiting and so on); (c) viscerosomatic reflexes causing vaso-

constriction in the skin, secretion of sweat (cold, pale, moist skin); (d) referred pains apparently radiating from the sternum.

The terminals of sympathetic nerves that are stimulated apparently are those in the ischemic region but evidence is conflicting as to whether these terminals are distributed throughout the muscle or limited to sheaths of the blood vessels affected. The nature of the stimuli may be chemical but this has not been established. The possibility also must be considered that stretching of the ventricular wall in the ischemic region may act as a mechanical excitant. Such an assumption would explain the amelioration of pain by reduction of arterial pressure and its intensification as pressure rises during decompensation.

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